Motor Nerve Conduction Velocity Determination in the Differential Diagnosis of Neuromuscular Disorders

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DETERMINATION of the motor nerve conduction velocity of peripheral nerves yields valuable diagnostic information not available through any other means. Although determination of motor nerve conduction velocity is of greatest value in differentiation of axonal disease from anterior horn cell disease, other valuable information may be obtained. For example, anomalous innervation can be traced; myopathic disease may be differentiated from disease of the peripheral nerves, upper motor neuron disease may be differentiated from lower motor neuron disease and localization of pathology along the course of the nerve may be done. Of course, electromyography alone may establish the presence of lower motor neuron disease. It may also differentiate between myopathic and neuropathic disease. There is difficulty in differentiation of anterior horn cell disease from disease of the axon with electromyography alone. Electromyography has been accepted in clinical medicine for over 12 years. Nerve conduction determination, although old to neurophysiology, is relatively new to clinical medicine. Its use as a clinical tool has paralleled the advances in electronics. Relatively simple apparatus has been developed which removes nerve conduction determinations from a complicated laboratory setting to a practical and useful clinical setting. It is the purpose of this paper to call to the attention of the profession this valuable clinical tool and to report our experience with it at the Tuskegee VA Hospital.

REVIEW OF LITERATURE

Hemholtz is reported to have first performed nerve conduction velocity determinations in man in the mid 19th Century. His apparatus was crude, but his results were similar to those found today. In 1927, Erlanger and Gasser[1, 2] published their findings of their study of nerves in the dog and bullfrog. Their study demonstrated the relationship between conduction velocity and the diameter of nerve fibers. Gasser and Grundfest[3] reported their study of nerve conduction velocity in relationship to the diameter of nerve fibers in rabbits and cats in 1939. They found that conduction velocity was approximately proportional to the axon diameter. Harvey and Masland[4] utilized peripheral nerve stimulation and recording of the action potential of muscles as an aid to diagnosis of myasthenia gravis. Their work, reported in 1945, demonstrated a progressive diminution of amplitude of the action potential of muscles in patients with this disease when the peripheral nerve supplying the muscle was stimulated with a supramaximal stimulus repetitively. Wagman and Lesse[5] reported on the relationship between the motor nerve conduction velocity of the ulnar nerve and the age of the person. Maximum conduction was found in young adults as early as the 4th or 5th year. In persons over 60 years of age, the conduction velocity was reduced by 10 per cent. Hodes, Larrabee and German[6] reported on their study of motor nerve conduction velocity in peripheral nerve injury and hysterical paralysis in 1948. They found that conduction velocity in peripheral nerve injuries was reduced during regeneration and related this to the fact that the diameter of the regenerating nerve was smaller than that of the original nerve. They found a progressive increase in conduction during the first year after repair. However, the conduction velocity never reached more than 60 per cent of the normal conduction velocity. Lambert and associates at the Mayo Clinic perform nerve conduction studies routinely on patients referred for electromyography. This is one

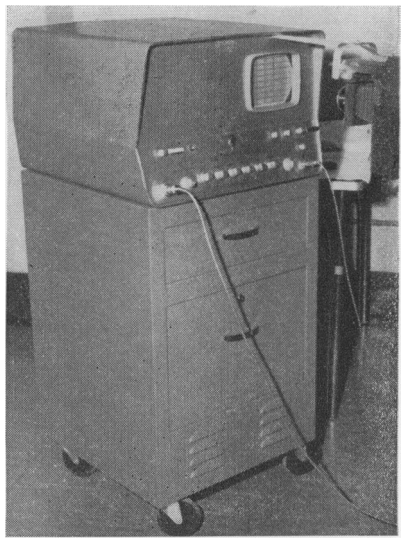

Fig. 1. Meditron Electromyograph, Model 201-A, with built in stimulus control unit. A Polaroid Land Camera is attached.

of the few centers where conduction velocity studies are done routinely on all referred for electromyography. Several reports have resulted from their work. In 1957 Eaton and Lambert[7] published a report of experience with electromyography and nerve conduction determination again emphasizing the value of nerve conduction velocity determination and electromyography in clinical medicine. More recently, Johnson and Olsen[8] reported on nerve conduction velocity determinations in normal individuals and some pathologic states. The clinical value of motor nerve conduction velocity determination was stressed.

APPARATUS

A Meditron, single channel electromyograph,

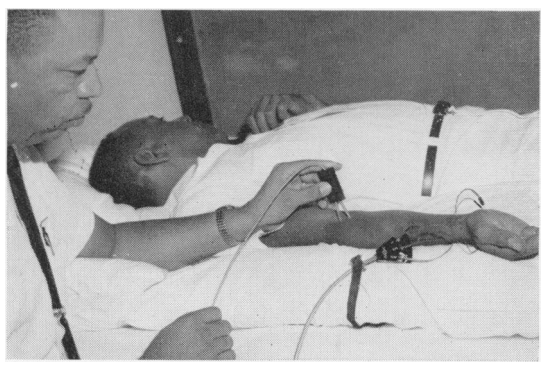

Fig. 2. Technique of percutaneous stimulation of the median nerve at the antecubital fossa. Recording electrode and ground electrode are shown in position in the opponens pollicis muscle.

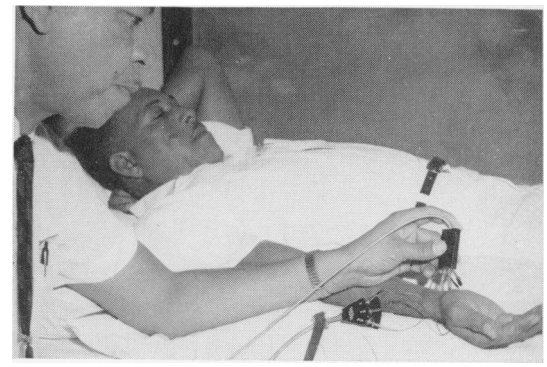

Fig. 3. Technique of percutaneous stimulation of the median nerve at the wrist. Again the recording and ground electrodes are in position in the opponens pollicis muscle.

Model 201-A, is used in our clinics for making electromyograms and motor nerve conduction studies. This unit has a stimulus control unit built in. It is designed to deliver a stimulating impulse during the brief sweep-fly back of the EMG oscilloscope. The duration of the stimulus is .05 milliseconds, and the intensity can be varied up to 350 volts. This is sufficient to excite virtually all motor axons. For practical purposes, the start of the sweep is the same as the stimulus artefact produced from the stimulus applied to the nerve. A Polaroid Land Camera is focused on the oscilloscope screen and a connecting cable attaches the camera to the stim-

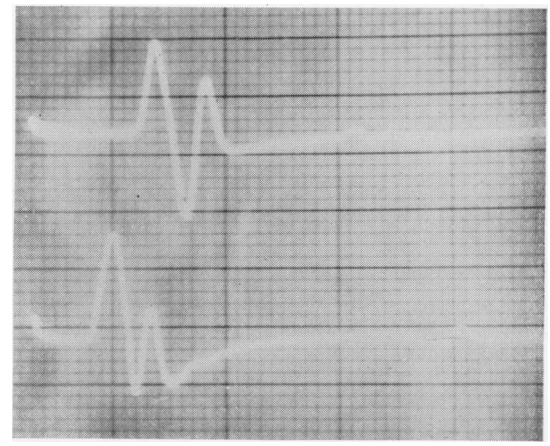

Fig. 4. Reproduction of a Polaroid photograph of the oscilloscope during examination of a normal median nerve. The top tracing shows the stimulus artefact on the left and response of the muscle on the right when the nerve was stimulated in the antecubital fossa. The bottom tracing represents the stimulus artefact and the response of the muscle when the nerve was stimulated at the wrist. Conduction time from antecubital fossa to wrist, as determined from the above tracing, is 4 milliseconds. Distance from antecubital fossa and wrist was 23.5 cm. Conduction velocity was 58.6 meters/sec., which is within normal limits.

ulus control unit. When the stimulus control unit is turned on, tripping the shutter lever of the camera will re-start the oscilloscope sweep, deliver a stimulus to the nerve, and record the response from the "pick-up" muscle. The apparatus is shown in Fig. 1.

TECHNIQUE

The nerve to be examined is stimulated at a proximal and a distal point along its course. Stimulation is percutaneous. Electrode paste is applied to the skin at the points of stimulation in order to lower skin resistance, thus necessitating less intensity of stimulus.

The most distal and accessible muscle supplied by the nerve is utilized in recording the response of the nerve to stimulation. The recording electrode is a coaxial intramuscular electrode, which is thrust into the belly of the muscle; a small intramuscular ground electrode is inserted proximal to the recording electrode. The coaxial and ground electrodes are connected to a pick-up cable which feeds the electrical responses into a preamplifier, amplifier, and then into a cathode ray oscilloscope and a loudspeaker. Surface electrodes may be used to pick up the electrical activity from the muscle. We have found that a more distinct "take-off" of the response of the muscle from the base line is obtainable when intramuscular rather than surface electrodes are used. This, in turn, means that the nerve conduction time may be determined more accurately. The sweep speed of the oscilloscope is set at 10 milliseconds per inch. The one inch graduations are re-divided into tenths with each tenth representing one of the small squares. When the oscilloscope is so calibrated, one of the small or one-tenth inch squares represents one millisecond. By counting the number of squares or fractions of squares between the stimulus artefact and the response of the muscle, the conduction time is determined in milliseconds. The conduction time between the distal point of stimulation and the response of the muscle is subtracted from the nerve conduction time between the proximal point of stimulation and the response of the muscle. This procedure eliminates the factors of end plate delay and the time required for the stimulus to spread through the muscle fibers. Thus, the nerve conduction time for the segment of nerve between the proximal and distal points of stimulation is obtained. The distance between the point proximally and distally is measured on the extremity with a steel tape or ruler. The distance in meters divided by the time in seconds will give the motor conduction of the nerve in meters per second. Figures 2 and 3 demonstrate the technique of stimulating the median nerve proximally and distally respectively. The intramuscular electrodes are demonstrated in the opponens pollicis muscle. Figure 4 demonstrates a photograph of the oscilloscope screen showing the stimulus artefact and the response of the muscle when the median nerve is stimulated proximally (top tracing) and distally (bottom tracing) in a normally conducting nerve. Figure 5 represents the same information in a diseased nerve.

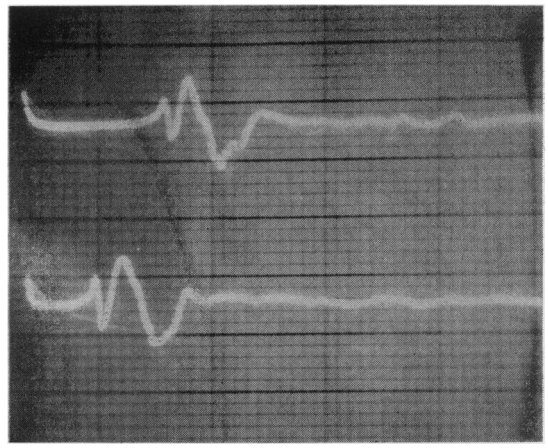

Fig. 5. Reproduction of a Polaroid photograph of the oscilloscope during examination of a diseased median nerve. Note that the conduction time from antecubital fossa to the opponens is delayed. Conduction time from antecubital fossa to wrist was 7.5 milliseconds. Distance from antecubital fossa to wrist was 27 cm. Conduction velocity was 36 meters per second, which is well below normal.

Four peripheral nerves lend themselves to easy examination. They are the ulnar, median, common peroneal and posterior tibial. The proximal point of stimulation of the ulnar nerve is in the ulnar groove at the elbow and just medial to the tendon of the flexor carpi ulnaris at the wrist. The recording electrode is placed on or in the belly of the abductor digiti quinti muscle. The median nerve is stimulated proximally in the antecubital fossa just medial to the pulsation of the brachial artery. The distal point of stimulation is at the wrist between the tendons of the palmaris longus and the flexor carpi radialis. The recording electrode is placed on or in the belly of the opponens pollicis muscle. The common peroneal nerve is

stimulated proximally at the lateral border of the popliteal fossa at the knee and at the ankle lateral to the tendon of the extensor hallucis longus (medial dorsal cutaneous branch). The recording electrode is placed in or on the belly of the extensor digitorum brevis muscle. Finally, the posterior tibial nerve is stimulated proximally at the inferior apex of the popliteal space in the midline and distally at the posterior border of the medial malleolus. The recording electrode is placed on or in the abductor digiti minimus of the foot.

DISCUSSION

We have found that normal motor nerve conduction velocity determined as outlined in preceding paragraphs, is 40 to 70 meters per second. The motor nerve conduction velocity varies within this range depending upon the nerve tested. The median nerve conducts fastest. Johnson and Olsen[8] reported the following mean values and standard deviations of motor nerve conduction velocities of various nerves in normal persons:

Peroneal	50.1 ± 7.2 meters/sec	(172 patients)
Median	53.1 ± 6.4 meters/sec	(68 patients)
Ulnar	55.1 ± 6.4 meters/sec	(67 patients)
Posterior tibial	50.2 ± 9.3 meters/sec	(20 patients)

They consider their variations slightly greater than values reported elsewhere and gave several possible reasons.

1. In the series reported, the determination was made on patients suspected of having motor unit disease.
2. Ages of the patients varied from 2 to 77 years with largest proportion in the range 2-10 years.
3. The conduction time was calculated between the stimulus artefact and the peak of the action potential instead of the "take-off" of the action potential.
4. Several different physicians performed the examinations.

Our values have been similar to those reported, though our series of patients is small as compared with those above. We feel that our findings are significant in that they agree substantially with those of other workers. When our series is larger, a subsequent report will be made with findings in normal and pathologic states.

Motor nerve conduction velocity is found to be reduced in polyneuritis, alcoholic neuropathy, diabetic neuropathy, the neural type of progressive muscular atrophy, Landry Guillain-Barré syndrome, localized neuropathy (carpal tunnel syndrome, tardy ulnar nerve palsy), and in regenerating nerves. In poliomyelitis, spinal type of progressive muscular atrophy and other diseases affecting the anterior horn cell body, the conduction velocity is normal or low normal. It averages 27 meters/sec in newborns and approaches normal by age 18 months to two years.[8] Motor nerve conduction is decreased in older persons. Wagman and Lesse[5] found that the maximum motor nerve conduction of ulnar fibers is present in young adults and as early as the fourth year. In persons over 60 years of age, they found the motor nerve conduction velocity to be reduced by 10 per cent. They alluded to metabolic, circulatory changes and temperature alterations as possible explanations. Finally, temperature affects nerve conduction. Increase in temperature of the part will cause an increase in motor nerve conduction velocity. A decrease in temperature of the part will cause a decrease in nerve conduction velocity.

SUMMARY

Motor nerve conduction velocity determination is proving to be a valuable clinical tool for investigation of the motor unit. Its principal value lies in its ability to distinguish axonal disease from disease of the anterior horn cell. Localization of a lesion along the course of the axon may be evidenced by a decreased conduction velocity when the nerve is stimulated above the lesion and a normal velocity when stimulated below the lesion. When combined with electromyography, even more information about the integrity of the motor unit is obtained. Pathology of the peripheral nerve reflects itself by a decrease in motor nerve conduction velocity below the limits of normal. A finding of a motor nerve conduction velocity below 40 meters per second is considered evidence of axonal involvement.

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ACKNOWLEDGEMENTS

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FOR YOUR CALENDAR

50th Annual Meeting
JOHN A. ANDREW CLINICAL SOCIETY
Tuskegee Institute, Ala.—April 8-13, 1962
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40th Annual Session
ASSOCIATION OF FORMER INTERNES AND RESIDENTS OF FREEDMEN'S HOSPITAL
Washington, D.C.—June 5-7, 1962

75th Annual Meeting
OLD NORTH STATE MEDICAL SOCIETY
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DIAMOND ANNIVERSARY CELEBRATION

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